

Supplementary tables

Table S1. Classification of Chromatin Regulatory Factors.

Gene name(a)	Description	Function(b)
Polycomb Repressive Complex 2		
<i>EZH2</i> *	Catalytic subunit	H3K27me1/me2/me3 HMT. Major role in stem cell identity maintenance. Also methylates GATA4. Interacts with DNMTs.
<i>SUZ12</i>	EZH2 coenzyme	Required for PRC2 H3K27 HMT activity (1). Interacts with SIRT1.
<i>EED</i>		Different isoforms determine PRC3 or PRC4 PRC2 variants.
<i>RBBP4 (RBAP46)*</i>		Required for the association of PRC2 to the histone tail (2). Binds Rb to regulate cell proliferation.
<i>RBBP7 (RBAP48)*</i>		Interacts with BRCA1 and may regulate cell proliferation and differentiation.
<i>PHF1 (PCL1)</i>		Mediates PRC2 intrusion into active H3K36 chromatin regions (3).
<i>PHF19 (PCL3)</i>		Mediates interaction of PRC2 with H3K36me3, essential for full PRC2 activity (4).
<i>ASXL1</i>		Associates with PRC2 to promote gene repression (5).
<i>MTF2 (PCL2)</i>		Required for PRC2-mediated Hox repression (6).
<i>JARID2 (JMJ)*</i>		Essential in embryonic development, inhibits H3K27me3 by PRC2.
<i>YY1*</i>		Interacts with PRC2, and it is required for EZH2-mediated H3K27me3 (7). Also part of chromatin remodelling INO80 complex.
<i>SIRT1*</i>	Class III HDAC	Transiently interacts with PRC2. Histone and protein deacetylase activity.
Polycomb Repressive Complex 1		
<i>EZH1</i>	Catalytic subunit	H3K27me1/me2/me3 HMT. Less critical for H3K27me3 formation than <i>EZH2</i> .
<i>BAP1</i>		Catalytic component of the PR-DUB complex, that specifically deubiquitinates H2AK119ub1.
<i>BM11</i>		Maintenance of transcriptional repression of key genes during development. H2AK119ub.
<i>RING1</i>		H2AK119ub.
<i>RNF2 (RING1B)</i>		H2AK119ub. Acts as the main ub ligase in PRC1.
<i>CBX2</i>		
<i>CBX3</i>		Part of PRC1-like complex 4 (8). Binds the nuclear lamina through lamin B receptor.
<i>CBX4</i>		
<i>CBX6</i>		
<i>CBX7</i>		Promotes H3K9me3. Regulates cellular lifespan by repressing CDKN2A.
<i>CBX8</i>		
<i>PCGF1 (NSPC1)</i>	BCOR complex	Represses CDKN1A expression in a RARE-dependent manner.
<i>PCGF2 (MEL18)</i>		
<i>PCGF6 (MBLR)</i>		
<i>PHC1</i>		
<i>PHC2</i>		
<i>PHC3</i>		
<i>AEBP2</i>		
<i>L3MBTL1</i>		Specifically recognizes me1 and me2 lysines.
Histone deacetylases		
<i>HDAC1</i> *		Controls embryonic stem cell differentiation (but not HDAC2) (9). Modulation of cell growth and apoptosis by down-regulation of p53. Also part of NuRD/Mi-2 ATP-dependent chromatin remodelling complex.
<i>HDAC2</i> *	Class I	Relevant role in haematopoiesis. Also part of NuRD/Mi-2 ATP-dependent chromatin remodelling complex.
<i>HDAC3</i>		Modulation of cell growth and apoptosis by down-regulation of p53.
<i>HDAC8</i>		
<i>HDAC4</i>		
<i>HDAC9</i>	Class IIa	Protects neurons from apoptosis.
<i>HDAC5</i>		
<i>HDAC7</i>		
<i>HDAC6</i>		
<i>HDAC10</i>	Class IIb	
<i>SIRT1</i> *	Class III, NAD-dependent	Interacts with PRC2, non-histone deacetylase activity. Involved in normal ageing through resistance to cellular stress. Deacetylates p53. Located in nucleus and cytoplasm (10).
<i>SIRT2</i>		Deacetylates alpha-tubulin. Located in the cytoplasm (10).
<i>SIRT3</i>		Located in the mitochondria (10).
<i>SIRT4</i>		

<i>SIRT5</i>		
<i>SIRT6</i>		Located in the nucleus (10). H3K9 and H3K56 deacetylase activity.
<i>SIRT7</i>		Located in the nucleus (10).
<i>HDAC11</i>	Class IV	
<i>ARID4A</i>		Bridging molecule to recruit HDACs.
<i>TBL1XR1</i>		Associates with HDAC3 (11).
<i>NCOR1</i>		Forms complex with HDAC1.
<i>TRIM28 (KAPI)*</i>		Proposed to be a transcriptional repressor. Mediates apoptosis through degradation of p53 (12).
Histone acetyltransferases		
<i>EP300</i>	Type A,	Acetylates all four core histones, and non-histone proteins like p53 and MyoD (13).
<i>CREBBP (CBP)</i>	CBP/P300 family	Critical role in embryonic development, acetylates both histone and non-histone proteins.
<i>NCOA3</i>		HAT activity not studied in detail.
<i>BRPF1 (TAF250)</i>	Type A	
<i>ATF2</i>		Specifically acetylates H2B and H4 <i>in vitro</i> .
<i>KAT6A (MOZ)</i>		Component of the MOZ/MORF complex, which has a histone H3 acetyltransferase activity.
<i>KAT6B (MORF)</i>		
<i>KAT5 (TIP60)</i>	Type A, MYST family	
<i>KAT8 (MOF)</i>		
<i>KAT7 (HBO1)</i>		Responsible for the bulk of histone H4 acetylation <i>in vivo</i> .
<i>KAT2A (GCN5)</i>	Type A, GNAT family	
<i>KAT2B (PCAF)</i>		
<i>HAT1</i>	Type B	
<i>ING4</i>		Facilitates targeting of HBO1-mediated acetylation to H3K4me3 sites (14).
<i>SET</i>	HAT inhibitor	Promotes apoptosis. Inhibits p300/CBP and PCAF-mediated acetyltransferase.
Histone methyltransferases		
<i>ASH1L (ASH1)</i>		H3K36 HMT.
<i>ASH2L</i>		H3K4 HMT. Complex with MLL
<i>ATF7IP (MCAF)*</i>		Required to stimulate SETDB1 activity, couples H3K9me3 with DNA methylation.
<i>DOT1L (KMT4)</i>		H3K79 HMT.
<i>EHMT2 (G9a)</i>		H3K9me1/me2, H3K27me HMT.
<i>EHMT1</i>		H3K9me1/me2 HMT.
<i>EZH2*</i>		H3K27me1/me2/me3 HMT. Major role in stem cell identity maintenance. Also methylates GATA4. Catalytic subunit of PRC2 complex.
<i>MEN1</i>		H3K4 HMT. Essential component of a MLL/SET1 HMT complex. Represses telomerase expression. Role in TGF β 1-mediated inhibition of cell-proliferation.
<i>MLL</i>		H3K4 HMT. Key regulator of development and hematopoiesis.
<i>MLL2</i>		
<i>MLL3</i>		H3K4 HMT.
<i>MLL4</i>		H3K4 HMT. Required to control the bulk of H3K4me3 during oocyte growth and preimplantation.
<i>MLL5</i>		H3K4me1/me2 HMT. Key regulator of hematopoiesis.
<i>NSD1 (KMT3B)</i>		H3K36, H4K20 HMT. May influence transcription positively or negatively.
<i>PRDM2 (RIZ1)</i>		H3K9 HMT.
<i>PRDM9</i>		H3K4me3 HMT. Essential for meiotic progression.
<i>RBBP5</i>		Complex with MLL.
<i>RTF1</i>		Required for H3K4me3 HMT on stem cell pluripotency genes.
<i>SETD1A (SET1A)</i>		H3K4 HMT.
<i>SETD1B (SET1B)</i>		H3K4 HMT.
<i>SETD2 (KMT3A)</i>		H3K36 HMT.
<i>SETD7 (SET7)</i>		H3K4 HMT.
<i>SETD8 (KMT5A)</i>		Trimethylates H4K20 (15).
<i>SETDB1 (ESET)</i>		H3K9 HMT.
<i>SETDB2</i>		H3K9 HMT.
<i>SMYD1</i>		H3K4 HMT (16).
<i>SMYD2 (KMT3C)</i>		H3K4me, H3K36me2 HMT. Also methylates TP53 and RB1.
<i>SMYD3</i>		H3K4me2/me3 HMT.
<i>SUV39H1 (KMT1A)</i>		H3K9me3 HMT, uses H3K9me1 as substrate.
<i>SUV39H2 (KMT1B)</i>		H4K20me3 HMT. Key in constitutive heterochromatin formation at pericentromeric regions.
<i>SUV420H1 (KMT5B)</i>		
<i>SUV420H2 (KMT5C)</i>		
<i>TRIM28 (KAPI)*</i>		Mediates silencing by recruiting SET1 H3K9me3 HMT and HDAC NuRD complex. Mediates apoptosis through degradation of p53 (12).

<i>WDR5</i>	Complex with MLL.
Histone demethylases	
<i>KDM1A (LSD1)*</i>	H3K4me2/me1, H3K9 HDM, also demethylates and stabilizes DNMT1.
<i>KDM1B (LSD2)*</i>	H3K4me2/me1 HDM. Required for <i>de novo</i> DNA methylation of a subset of imprinted genes during oogenesis.
<i>KDM2A</i>	H3K36me2 HDM. Required to maintain heterochromatic state at centromeres.
<i>KDM2B</i>	H3K4me3, H3K36me2 HDM. Represses rRNA genes.
<i>KDM3A</i>	H3K9me2/me1 HDM.
<i>KDM3B</i>	H3K9 HDM.
<i>KDM4A</i>	H3K9me3, H3K36me3 HDM.
<i>KDM4B</i>	H3K9me3 HDM.
<i>KDM4C</i>	H3K9me3, H3K36me3 HDM.
<i>KDM4D</i>	H3K9me3/me2 HDM.
<i>KDM5A (RBP2)</i>	H3K4me2/me3 HDM. Prominent role in cell differentiation and senescence (17).
<i>KDM5B (PLUI)</i>	H3K4me3/me2/me1 HDM.
<i>KDM5C (SMCX)</i>	H3K4me3/me2 HDM. Participates in the repression of neuronal genes.
<i>KDM5D (SMCY)</i>	H3K4me3/me2 HDM.
<i>KDM6A (UTX)</i>	H3K27me2/me3 HDM. Regulation of HOX gene expression.
<i>KDM6B (JMJD3)</i>	H3K9me2, H3K27me2, H4K20me1 HDM. Required for brain development.
<i>JHDM1D (KDM7A)</i>	H3K36me2 HDM. Required for G2/M cell cycle progression.
<i>KDM8 (JMJD5)</i>	H3K9 HDM.
<i>JMJD1C (TRIP8)</i>	H3R2, H4R3 HDM. Key regulator of haematopoietic differentiation.
<i>JMJD6</i>	H3K9me2 HDM.
<i>PHF2</i>	H3K9me1/me2, H3K27me2, H4K20me1 HDM. Key role in cell cycle progression.
<i>PHF8</i>	H3K27me3/me2/me1 HDM (18).
<i>UTY</i>	
<i>JARID2 (JMJ)*</i>	Essential role in embryonic development, inhibits PRC2 trimethylation of H3K27 (19).
DNA methyltransferases	
<i>DNMT1</i>	Maintainins methylation patterns established in development.
<i>DNMT3A</i>	Genome-wide <i>de novo</i> methylation, essential for the establishment of DNA methylation patterns during development.
<i>DNMT3B</i>	
<i>DNMT3L</i>	Catalytically inactive, but essential for DNMT3A and DNMT3B function.
<i>MECP2</i>	
<i>MBD1</i>	Essential for embryonic development. Specifically bind methylated DNA and repress transcription at methylated promoters.
<i>MBD2*</i>	
<i>MBD4</i>	
<i>ATF7IP (MCAF)*</i>	Mediates MBD1 transcriptional repression, couples H3K9me3 with DNA methylation.
<i>KDM1A (LSD1)*</i>	HDM, also demethylates and stabilizes DNMT1.
<i>KDM1B (LSD2)*</i>	HDM, required for <i>de novo</i> DNA methylation of a subset of imprinted genes during oogenesis.
DNA demethylases	
<i>TET1</i>	Converts 5mC to 5hmC
<i>TET2</i>	Putative role in DNA demethylation (20).
<i>AICDA (AID)</i>	May play a role in DNA demethylation.
<i>TDG</i>	Essential for DNA demethylation (21).
ATP-dependent chromatin remodelling	
<i>SMARCA2 (BRM)</i>	SWI/SNF complex is required for transcriptional activation of genes normally repressed by chromatin (22).
<i>SMARCA4 (BRG1)</i>	Catalytic component of SWI/SNIF complex (23). Essential for the maintenance of multipotent neural stem cells.
<i>SMARCB1 (BAF47)</i>	
<i>SMARCC1</i>	
<i>SMARCC2</i>	
<i>SMARCD1</i>	
<i>SMARCD2</i>	
<i>SMARCD3</i>	
<i>SMARCE1 (BAF57)</i>	
<i>ARID1A</i>	
<i>ARID1B (BAF250B)</i>	Required for the stability of the SWI/SNF chromatin remodelling complex SWI/SNF-B.
<i>ARID2 (BAF200)</i>	Required for maximal SMARCA4 activity and for the association of the SWI/SNF complex with chromatin.
<i>ACTL6A (BAF53A)</i>	
<i>ACTL6B (BAF53B)</i>	
<i>DPF1 (BAF45B)</i>	
<i>DPF2 (BAF45D)</i>	

<i>DPF3 (BAF45C)</i>		
<i>EP400</i>		Regulates nucleosome stability during DNA repair (24).
<i>PBRM1</i>		Regulator of cell proliferation.
<i>PHF10 (BAF45A)</i>		Required for the proliferation of neural progenitors.
<i>MTA1</i>		
<i>MTA2</i>		
<i>MTA3</i>		Maintenance of the normal epithelial architecture through the repression of SNAI1 transcription in a HDAC-dependent manner.
<i>CHD3 (Mi-2α)</i>	NuRD/Mi-2 complex has	
<i>CHD4 (Mi-2β)</i>	ATP-dependent chromatin remodelling	Main component of the NuRD/Mi-2 complex.
<i>GATAD2A</i>		
<i>GATAD2B</i>		
<i>HDAC1*</i>	activity and HDAC activity	
<i>HDAC2*</i>		
<i>MBD2*</i>		Essential for embryonic development. Also bind methylated DNA.
<i>RBBP4 (RBAP46)*</i>		Also part of PRC2 complex.
<i>RBBP7 (RBAP48)*</i>		
<i>INO80</i>	INO80 complex has DNA- and nucleosome-activated ATPase activity and catalyzes ATP-dependent nucleosome sliding (25).	
<i>TFPT</i>		Putative regulatory component of the INO80 complex
<i>YY1*</i>		Also interacts with PRC2 and is required for EZH2-mediated H3K27me3 (7).
<i>SMARCA1 (SNF2L)</i>		
<i>SMARCA5 (SNF2H)</i>	ISWI complex mobilizes mononucleosome	Required for replication of pericentric heterochromatin in S-phase specifically in conjunction with BAZ1A.
<i>BAZ1A (ACFI)</i>		
<i>BAZ1B (WSTF)</i>	s away from DNA ends	Acts as a mark that distinguishes between apoptotic and repair responses to genotoxic stress. Maintenance of chromatin structures during DNA replication processes.
<i>BAZ2A (TIP5)</i>	without changing the arrangement	
<i>BPTF</i>	of DNA on the surface of the histone octamer (22).	Binds H3K4me3.
<i>CHRAC1</i>		
<i>POLE3</i>		
<i>RSF1</i>		
<i>RBBP4 (RBAP46)*</i>		Also part of PRC2 complex.
<i>RBBP7 (RBAP48)*</i>		
<i>CHD1</i>		Required for the maintenance of open chromatin and pluripotency in ESC.
<i>CHD2</i>		SNF2-related helicase/ATPase domains.
<i>HNF1A</i>		Possible regulation of transcription through chromatin remodelling (26).
<i>IKZF1*</i>		Targets NuRD/Mi-2 and SWI/SNF complexes in a single complex.
Global chromatin regulators		
<i>LMNA</i>	lamin A/C	
<i>LMNB1</i>	lamin B1	Global heterochromatic changes induced by lamin perturbation are often mirrored by altered levels of chromatin-associated epigenetic histone marks (27).
<i>LMNB2</i>	lamin B2	
Other chromatin regulators		
<i>BAG6</i>	Complex EP300	p300-mediated p53 acetylation upon DNA damage. May mediate H3K4me2.
<i>ATRX</i>	ATRX-DAXX complex	Thought to regulate deposition of H3.3 at heterochromatic regions of the genome, including telomeres (28).
<i>DAXX</i>		
<i>MUM1</i>		Opens chromatin to facilitate DNA damage repair (29).

*Genes with more than one function in chromatin remodelling appear more than once in the table.

(a) HGNC HUGO gene names. In parenthesis, common alternative gene names.

(b) Gene function provided by Uniprot, unless otherwise stated.(30)

Table S2. Described oncogenic alterations in Chromatin Regulatory Factors. This is an exhaustive compilation of alterations(*) reported in CRFs not included in Table 1. Gene names correspond to HUGO HGNC approved symbols. In bold typeface, genes included in the Cancer Gene Census (CGC) (31). ALL: Acute Lymphocytic Leukaemia; AML: Acute Myeloid Leukaemia; B-ALL: B Acute Lymphoblastic Leukaemia; B-NHL: B-cell non-Hodgkin Lymphoma; CLL: Chronic Lymphocytic Leukaemia; ccOC: Clear Cell Ovarian Carcinoma; ccRCC: clear-cell Renal Cell Carcinoma; CMML: Chronic Myelomonocytic leukaemia; ESCC: Oesophageal Squamous Cell Carcinoma; FL: Follicular Lymphoma; HCC: Hepatocellular Carcinoma; HL: Hodgkin Lymphoma; HNSCC: Head and Neck Squamous Cell Carcinoma; MCL: Mantle cell Lymphoma; MDS: Myelodysplastic Syndrome; MSI: Microsatellite instability; NMSC: Non-Melanoma Skin Cancer; NSCLC: Non-Small Cell Lung Carcinoma; OSCC: Oral Squamous Cell Carcinoma; RCC: Renal Cell Carcinoma; T-ALL: T Acute Lymphoblastic Leukaemia.

*Evidence based solely on cancer cell lines is excluded from this table. Only evidence in human samples have been used. Effects of pharmacological inhibition are not included. Germline polymorphisms are excluded.

Gene	Literature evidence
<i>AEBP2</i>	Deleted in AML (32).
<i>ATF2</i>	Over-expressed in melanoma (33).
<i>BAZ1A</i>	Amplified in ESCC (34). Deleted in papillary type 2 RCC (35).
<i>BMII</i>	Over-expressed in B-NHL, leukaemia, MCL, medulloblastoma, neuroblastoma, NSCLC (36) and prostate tumours (37).
<i>CBX2</i>	Over-expressed in breast cancer (38).
<i>CBX3</i>	Over-expressed in osteosarcoma (39), myxoid liposarcoma, colon, breast, esophageal, cervical, and lung tumours (40).
<i>CBX7</i>	Over-expressed in lymphoma (41). Down-regulated in bladder (42), and aggressive gastric (43), pancreatic (44) and thyroid cancer (45).
<i>CHD1</i>	Mutated in high MSI gastric and colorectal cancers (46). Deleted in prostate cancer (47).
<i>CREBBP</i>	Mutated in AML, ALL, DLBCL, N-NHL (CGC), bladder (48), medulloblastoma (49) and SCLC (50). LOH in lung (51).
<i>DAXX</i>	Mutated in paediatric glioblastoma and neuroendocrine pancreatic tumours (CGC). Over-expressed in prostate cancer (52).
<i>DNMT1</i>	Over-expressed in AML (53), gliomas (54) and pancreatic tumours (55).
<i>DNMT3B</i>	Over-expressed in breast (56), colorectal and stomach (57), prostate cancer (58), advanced stages of DLBCL (59).
<i>DNMT3L</i>	Over-expressed in testicular embryonal carcinoma (60). Loss of methylation and consequent over-expression in cervical cancer (61).
<i>EHMT2</i>	Over-expressed in bladder (62), resistant cervical (63) and aggressive lung tumours (64).
<i>EPC1</i>	Mutated in pancreatic cancer (65).
<i>EZH1</i>	Over-expressed and amplified in myeloproliferative neoplasms (66).
<i>EZH2</i>	Mutated in DLBCL (CGC), MDS (67). Over-expressed in bladder, breast, colon, liver, melanoma and prostate tumours; DLBCL, HL and MCL (36).
<i>GATAD2B</i>	Deleted in OSCC (68).
<i>HDAC1</i>	Over-expressed in HCC (69). Down-regulated in aggressive breast tumours (70).
<i>HDAC2</i>	Mutated in colon cancer with microsatellite instability (71). Over-expressed in gastrointestinal tumours (72), prostate (73), aggressive HCC (74), lung (75), cervical (76), ovarian and endometrial endometrioid carcinomas (77).

<i>HDAC3</i>	Over-expressed in gastrointestinal tumours (72), b-cell lymphomas (78) and CLL (79).
<i>HDAC4</i>	Mutated in melanoma (80) and breast cancer (81). Over-expressed in T-ALL (82) and treatment-resistant ovarian tumours (83).
<i>HDAC5</i>	Over-expressed in B-ALL (82) and aggressive medulloblastoma (84).
<i>HDAC6</i>	Over-expressed in HCC (85), cisplatin-resistant NSCLC (86) and breast tumours with good prognosis (87). Down-regulated in CLL (79).
<i>HDAC7</i>	Over-expressed in pancreatic adenocarcinoma (88) and aggressive childhood ALL (82).
<i>HDAC8</i>	Over-expressed in aggressive neuroblastoma (89).
<i>HDAC9</i>	Over-expressed in high grade medulloblastoma (84) and childhood ALL with poor prognosis (82). Amplified in OSCC (68).
<i>HDAC10</i>	Down-regulated in adrenocortical tumours (90), CLL (91) and aggressive NSCLC (92).
<i>HNF1A</i>	Mutated in neuroendocrine tumours (93), endometrial cancer (94), high MSI CRC (95) and hepatocellular adenoma (96). Down-regulated in aggressive HCC (97).
<i>IKZF1</i>	Mutated in ALL, DLBCL (CGC). Deleted in aggressive paediatric B-ALL (98).
<i>ING4</i>	Down-regulated in HNSCC (99), melanoma (100), gastric adenocarcinoma (101), lung tumours (102) and colorectal cancer (103). Deleted in HNSCC (99) and breast tumours (104).
<i>JARID2</i>	Mutated in NSCLC (105). Deleted in AML (32).
<i>JMJD1C</i>	Over-expressed in pancreatic ductal adenocarcinoma (106).
<i>JMJD6</i>	Over-expressed in aggressive breast tumours (107).
<i>KAT5</i>	Down-regulated in gastric cancer (108), aggressive melanoma (109) and advanced colorectal carcinoma (110).
<i>KAT6A</i>	Translocated in AML (111).
<i>KAT6B</i>	Translocated in AML (111) and benign uterine tumours (112).
<i>KAT7</i>	Over-expressed in testicular, breast, ovarian, bladder, oral and oesophageal carcinomas (113).
<i>KAT8</i>	Down-regulated in breast carcinoma and medulloblastoma (114).
<i>KDM1A</i>	Over-expressed in NSCLC (115), highly malignant sarcomas (116), bladder (117) and aggressive prostate tumours (118). Down-regulated in breast carcinoma (119).
<i>KDM2A</i>	Down-regulated in prostate cancer (120).
<i>KDM2B</i>	Over-expressed in ALL, AML (121) and pancreatic ductal adenocarcinoma (106).
<i>KDM3A</i>	Over-expressed in prostate cancer (122) and RCC (123).
<i>KDM3B</i>	Over-expressed in ALL (124) and prostate cancer (122).
<i>KDM4A</i>	Over-expressed in breast (125) and prostate cancer (122). Down-regulated in bladder tumours (126).
<i>KDM4B</i>	Over-expressed in gastric cancer (127).
<i>KDM4C</i>	Over-expressed and amplified in breast cancer (128).
<i>KDM5A</i>	Mutated in AML (CGC). Down-regulated in melanoma (129). Over-expressed in breast tumours with good prognosis (130) and in pancreatic ductal adenocarcinoma (106).
<i>KDM5B</i>	Over-expressed in breast tumours, prostate cancer (122) and uveal melanoma (131).
<i>KDM6B</i>	Over-expressed in HL (132) and pancreatic ductal adenocarcinoma (106).
<i>LMNA</i>	Over-expressed in aggressive colorectal cancer (133). Down-regulated in DLBCL (134), ALL and NHL (135).
<i>LMNB1</i>	Over-expressed in HCC (136) and colorectal tumours (137).
<i>MBD4</i>	Mutated in sporadic colon cancer (138) and HNPCC with MSI (139).

<i>MECP2</i>	Over-expressed in breast tumours (140).
<i>MEN1</i>	Mutated in pancreas, parathyroid (CGC) and in lung carcinoids (141). MLL-fusion partner in leukaemias (142).
<i>MLL5</i>	Down-regulated in poor prognosis AML (143).
<i>MTA1</i>	Over-expressed in OSCC, ESCC, early NSCLC, HCC, osteosarcoma, and colorectal, pancreatic, endometrial, ovarian, prostate, breast and gastric cancers. It is one of the most commonly over-expressed genes in human tumours (144).
<i>MTA2</i>	Over-expressed in NSCLC (145), aggressive HCC (146) and epithelial ovarian cancer (147).
<i>MUM1</i>	Over-expressed in aggressive PCLBCL (148) and CLL (149), DLBCL and HL (150).
<i>NCOA3</i>	Over-expressed in HCC, breast (151), urothelial carcinoma of the bladder (152), NSCLC (153) and prostate tumours (154). Amplified in breast cancer (155). Fusion partner of KAT6A in AML (156).
<i>PCGF2</i>	Over-expressed in aggressive medulloblastoma (157). Down-regulated in breast tumours (158) and high-grade prostate cancer (159).
<i>PHC1</i>	Over-expressed in ALL (36).
<i>PHF8</i>	Over-expressed in prostate cancer (122).
<i>PHF19</i>	Over-expressed in colon, skin, lung, rectal, cervical, uterine and hepatic tumours (36).
<i>PRDM2</i>	Mutated in endometrial, gastrointestinal (160) and colon tumours with MSI (161), melanoma (162). Over-expressed in ALL (163). Down-regulated in ESCC (164), neuroblastoma (165), HCC (166), epithelial ovarian carcinoma (167), thyroid carcinoma (168) and AML (163). Deleted in parathyroid tumours (169).
<i>RBBP4</i>	Over-expressed in HPV-positive oropharyngeal tumours (170). Down-regulated in mucoepidermoid carcinoma (171).
<i>RBBP5</i>	Amplified in glioblastomas (172).
<i>RBBP7</i>	Over-expressed in NSCLC (173) and breast tumours (174).
<i>RING1</i>	Over-expressed in prostate tumours (37).
<i>RSF1</i>	Over-expressed in NSCLC (175), urinary bladder (176), colon (177), gallbladder (178), nasopharyngeal (179) and ovarian aggressive carcinomas (180). Amplified in aggressive ovarian carcinoma (181).
<i>SET</i>	Mutated in AML (CGC). Over-expressed in colorectal adenocarcinoma (182) and paediatric B-ALL and T-ALL (183).
<i>SET8</i>	Over-expressed in aggressive breast tumours (184).
<i>SETDB2</i>	Deleted in CLL (185).
<i>SIRT1</i>	Over-expressed in leukaemia, prostate, skin and colon cancers (186) Down-regulated in breast tumours and HCC (187).
<i>SIRT2</i>	Down-regulated in gliomas (188).
<i>SIRT3</i>	Down-regulated in HCC (189).
<i>SIRT6</i>	Down-regulated in pancreas and colorectal cancer (190). Deleted in colorectal cancer (190).
<i>SIRT7</i>	Over-expressed in breast (191) and thyroid carcinoma (192).
<i>SMARCB1</i>	Mutated in malignant rhabdoid tumours (CGC).
<i>SMARCC1</i>	Over-expressed in prostate cancer (193) and precancerous cervical lesions (194). High expression correlates with good prognosis in colorectal cancer (195).
<i>SMARCD1</i>	Mutated in breast tumours (196).
<i>SMARCD3</i>	Over-expressed in advanced neuroblastoma (197).
<i>SMARCE1</i>	Over-expressed in aggressive endometrial carcinoma (198).
<i>SMYD2</i>	Over-expressed in ESCC (199).
<i>SMYD3</i>	Over-expressed in colorectal cancer (200).
<i>SUZ12</i>	Mutated in endometrial stromal tumours (CGC). Over-expressed in breast, colon, liver (36) and ovarian tumours (201).

	Amplified in MCL (202).
<i>TBL1XR1</i>	Over-expressed in SCC (203). Deleted in ALL (204) and PCNSL (205).
<i>TET1</i>	Mutated in T-ALL (206). Down-regulated in prostate and breast tumours (207).
<i>TFPT</i>	Mutated in pre-B ALL (CGC).
<i>TRIM28</i>	Over-expressed in colorectal tumours (208), gastric cancer cell lines (209), NSCLC and breast (210). Over-expression predicts better survival in early lung tumours (210). High expression indicates good prognosis in gastric cancer (209).
<i>YY1</i>	Over-expressed in prostate, colon, ovary, breast, bone, liver, lung, bladder, cervix, skin and blood (DLBCL, AML, CML, ALL, HL, BL, MCL, CLL and FL) cancers (211). Down-regulated in melanomas, paediatric osteosarcomas and urothelial carcinomas (211). There are contradictory results on the prognostic significance of YY1 in cancer (211).

Table S3. Mutually exclusivity test for mutations in genes coding proteins that act in the same complex.

Protein complex	Site	Gene 1	Gene 2	P value Fisher Test
SWISNF	Bladder	ARID2	ARID1A	0.02
ISWI	Bladder	BAZ2A	BPTF	0.03
NURDMI2	Bladder	CHD3	CHD4	$P < 10^{-16}$
SWISNF	Breast	ARID2	ARID1A	0.01
SWISNF	Breast	SMARCA2	ARID1A	0.01
SWISNF	Breast	SMARCA4	ARID1A	$P < 10^{-16}$
ISWI	Breast	BAZ2A	BPTF	$1.08 * 10^{-5}$
NURDMI2	Breast	CHD3	CHD4	$1.13 * 10^{-6}$
PRC1	Breast	BAP1	PHC3	$P < 10^{-16}$
SWISNF	Head & Neck	ARID2	SMARCA4	0.05
NURDMI2	Head & Neck	CHD3	CHD4	$1.98 * 10^{-5}$
PRC1	Head & Neck	BAP1	PHC3	0.01
SWISNF	Lung	SMARCA2	SMARCA4	0.04
SWISNF	Lung	SMARCA2	ARID1A	0.04
ISWI	Lung	BAZ2A	BPTF	$P < 10^{-16}$
NURDMI2	Lung	CHD3	CHD4	$8.35 * 10^{-9}$
PRC1	Lung	BAP1	PHC3	0
SWISNF	Ovary	ARID2	SMARCA4	0.05
SWISNF	Uteri	PBRM1	ARID1A	0.02
SWISNF	Uteri	SMARCA4	ARID1A	0
ISWI	Uteri	BAZ2A	BPTF	0
NURDMI2	Uteri	CHD3	CHD4	$5.29 * 10^{-8}$

Table S4. Gene regulatory modules collected for the analysis.

Group	Name	Cell type	Nº of genes	Source
EP300	EP300 ES	ES	1191	Lister <i>et al.</i> 2009 (212)
	EP300 CD4	CD4	3792	Wang <i>et al.</i> 2009 (213)
Activating histone marks	H3K4me3 ES	ES	12312	ENCODE (214)
	H3K4me3 CD4	CD4	11423	Barski <i>et al.</i> 2007 (215)
	H3K4me3 gm12878	gm12878	11771	ENCODE (214)
	H3K9ac ES	ES	10489	ENCODE (214)
	H3K9ac CD4	CD4	6906	Wang <i>et al.</i> 2009 (213)
	H3K9ac gm12878	gm12878	9918	ENCODE (214)
Repressive histone marks	H3K27me3 ES	ES	6665	ENCODE (214)
	H3K27me3 CD4	CD4	5207	Wang <i>et al.</i> 2009 (213)
	H3K27me3 gm12878	gm12878	6099	ENCODE (214)
Replication Timing	Late RT ES	ES	918	Hansen <i>et al.</i> 2010 (216)
	Late RT lymphoid	lymphoid	260	Hansen <i>et al.</i> 2010 (216)

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